

Ventricular Opacification During Carotid Angiography Secondary to Rupture of Intracranial Aneurysm

Case Report¹

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ABSTRACT—Rupture of intracranial aneurysms during cerebral angiography is a rare occurrence. A case of such a rupture with extravasation of contrast material into the cerebral ventricular system and onto the floor of the middle cranial fossa is presented. The role of angiography as a possible causative factor is discussed.

INDEX TERMS: Aneurysm, intracranial • Brain, hemorrhage • Cerebral Angiography, complications • Cerebral Blood Vessels, rupture • Meninges, hemorrhage

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SINCE HEMORRHAGE into the cerebral ventricles was described by Sanders (15) in 1881, there have been two reports (5, 7) of an unequivocal nonoperative diagnosis *i.e.*, demonstration of extravasation of contrast material into the ventricular system during cerebral angiography. These cases resulted from rupture of congenital aneurysms but others report rupture of congenital aneurysms with extra-

ventricular extravasation during cerebral angiography (8-10, 13, 18).

In this paper, we present a case of rupture of an intracranial aneurysm during carotid angiography and comment on the role of angiography as a possible causative factor. To our knowledge, this will be the third such case in the literature.

The classic picture of intraventricular hemorrhage as described by Sanders (15) and McDonald (12) consists of acute onset of persistent coma, symptoms of midbrain herniation, decerebration in cases of long survival, meningismus, early persistent central regulation disorders, and death usually within 48 hours. This classic picture does not always occur as several nonlethal episodes of intraventricular hemorrhage have occurred in patients examined in our department during the past three years.

Pia (14) classified intraventricular bleeding into three morphologic types. The first is termed total hemocephaly in which the entire ventricular system is filled with clot or hematoma. The second is partial hemocephaly in which blood masses fill circumscribed portions of the ventricular system, usually a lateral ventricle, but rarely both lateral ventricles. Associated intracerebral hematoma is usually present in these types. In the third type, simply termed intraventricular hemorrhage, fresh blood is present in the cerebrospinal fluid without evidence of a hematoma. Pia's classification has prognostic significance in that the mortality rates were 100% with total hemocephaly, 56% with partial hemocephaly and 14% with intraventricular hemorrhage. The great majority of survivors underwent surgical intervention.

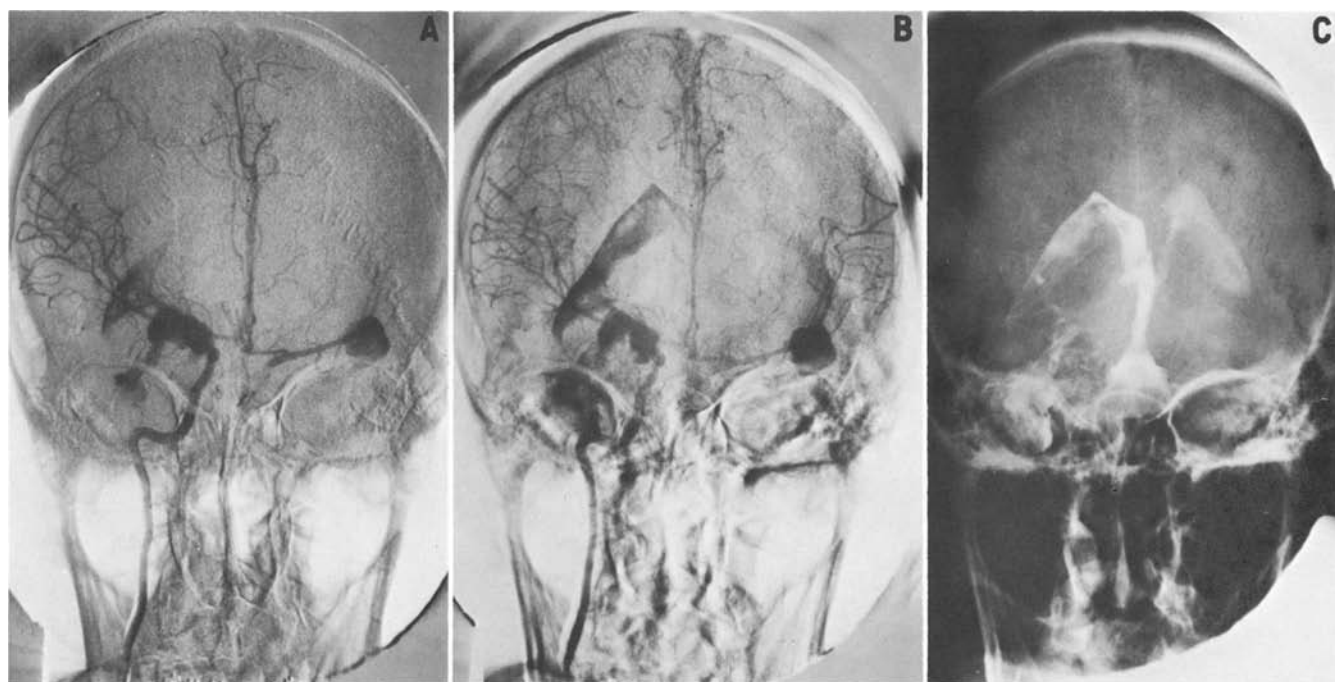


Fig. 1. A. Anteroposterior view of selective right internal carotid arteriogram exposed 1/2 second after initiation of injection demonstrates spontaneous opacification of the left anterior, middle and internal carotid arteries, small caliber of the extradural portion of the right internal carotid artery, small caliber of major intracranial vessels bilaterally, left middle cerebral artery aneurysm, large lobulated aneurysm near the bifurcation of the right internal carotid artery, elevation of the proximal right middle cerebral artery, and extravasation of contrast material into the right lateral ventricle and right middle cranial fossa.

B. Anteroposterior view exposed 1/2 second after Figure 1, A demonstrates increased extravasation into the right middle cranial fossa and increased opacification of the right lateral ventricle.

C. Anteroposterior view exposed 4 1/2 seconds after Figure 1, A showing opacification of essentially the entire ventricular system and extravasated contrast material in the right middle cranial fossa.

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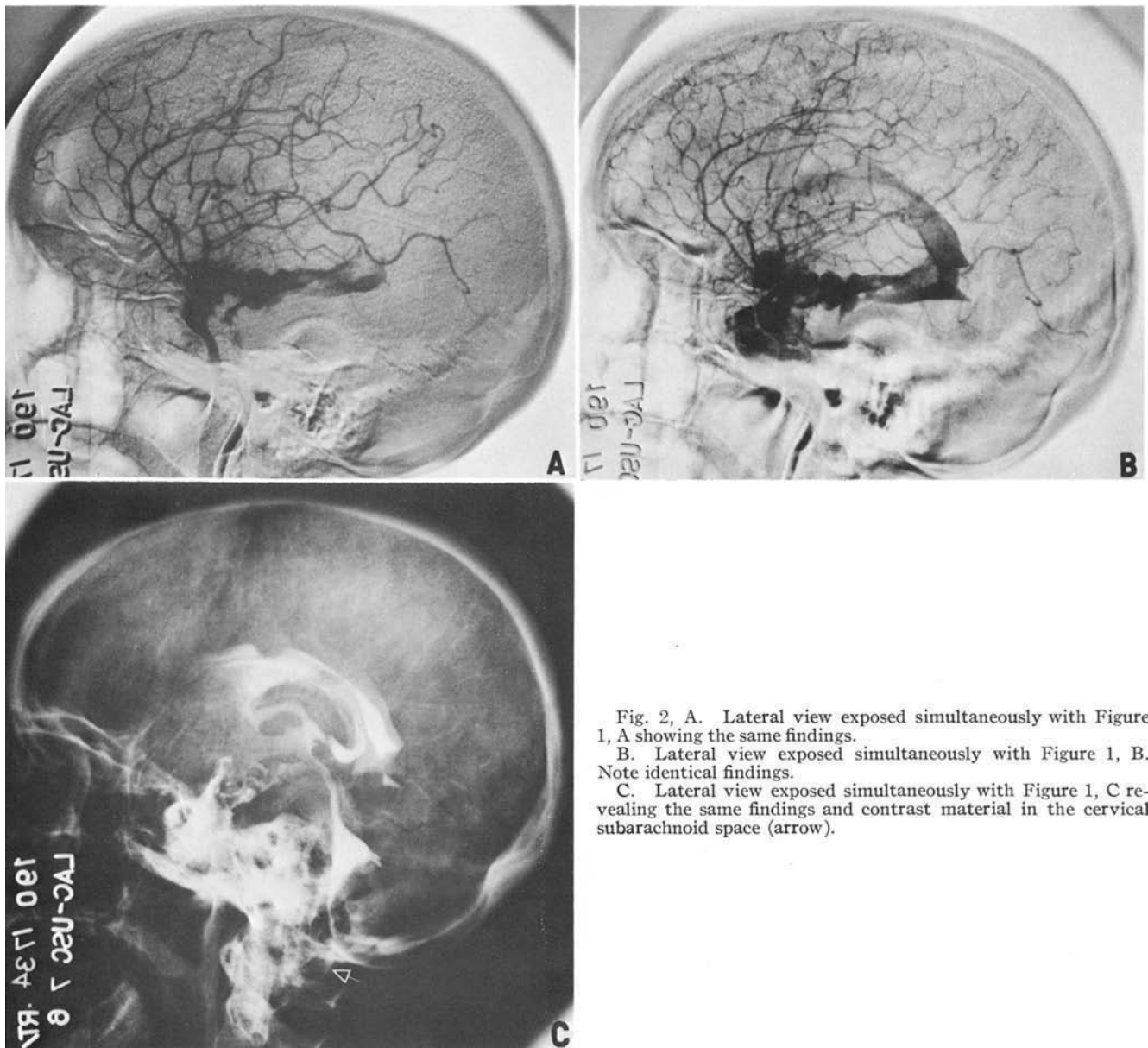


Fig. 2, A. Lateral view exposed simultaneously with Figure 1, A showing the same findings.

B. Lateral view exposed simultaneously with Figure 1, B. Note identical findings.

C. Lateral view exposed simultaneously with Figure 1, C revealing the same findings and contrast material in the cervical subarachnoid space (arrow).

CASE REPORT

A 41-year-old Caucasian woman was transferred to Los Angeles County-University of Southern California Medical Center on July 6, 1971 from an emergency hospital where she was taken after her car hit a utility pole. When first seen at the emergency hospital, she was alert and oriented but shortly afterwards suffered a grand mal seizure and was transferred to LAC/USC Medical Center.

On arrival, she was agitated and delirious with bilateral, equally dilated, poorly reactive pupils; left hemiparesis; and a left central VII nerve palsy. Right carotid angiography was requested to confirm or exclude the presence of an epidural or subdural hematoma.

Immediately following the injection of 10 ml of Conray at 125 PSI, the patient lapsed into a grand mal seizure with immediate coma. Examination of the biplane films (Figs. 1, and 2) revealed inadvertent cannulation of a spastic right internal carotid artery with visualization of spastic middle and anterior cerebral arteries bilaterally and supracaloid left internal carotid artery. A large aneurysm of the left

middle cerebral artery, a large ruptured aneurysm near the bifurcation of the right internal carotid with extravasation of media into the lateral ventricle and the floor of the right middle cranial fossa, and an avascular right temporal lobe mass, indicating recent intracerebral bleeding, were demonstrated. Within three seconds of injection, the entire ventricular system was visualized: contrast material had passed into the cervical subarachnoid space.

The patient never regained consciousness and died 71.5 hours later.

DISCUSSION

Based on the limited studies of Greitz (6) and Bakay and Sweet (2), most cerebral angiographers, including ourselves, feel that no significant increase in intracarotid pressure occurs during carotid angiography. Greitz's results were primarily based on 4-ml injections and Bakay and Sweet's on 10-12 ml, with injection times of 2-3 seconds. Neither of these are comparable to the routine situation in our department where 10-ml common carotid artery and 6-8 ml internal carotid

artery injections are routinely done in 1.0–1.5 seconds utilizing Taveras Picker, Turner MDT and Viamonte-Hobbs injectors. We feel that the volumes and injection times utilized in our department are probably comparable to those of most neuroradiology departments.

Lin *et al.* (11) reported that slight elevation (5–25 mm Hg) of intracarotid systolic pressure occurred in 7 of 12 patients during retrograde brachial injections of 45 ml by hand and at 225 and 625 PSI by pressure injector. This at least suggests that slight elevation of intracarotid pressure may occur during carotid angiography.

During selective vertebral angiography utilizing forceful hand injections of 5–6 ml of contrast material and small catheters with tips at the level of the 6th cervical vertebra, there is an extremely high incidence of retrograde filling of the opposite vertebral artery. This can logically be explained only in terms of a transient increase in intravertebral artery pressure. A similar change in the internal carotid artery is certainly not inconceivable.

Additional evidence which suggests that elevation of intracarotid pressure may occur during carotid angiography is the increased incidence of transient opacification of the basilar and contralateral proximal anterior cerebral arteries during selective internal carotid angiography compared to common carotid angiography.

The elasticity of arteries and the large total volume of the arterial system accounts for most, if not all, of the stability of the intraluminal pressure following forceful injections of relatively small volumes of solutions. In view of the seemingly conflicting reports of Greitz, and Bakay and Sweet in comparison with Lin and associates, we feel that additional investigations of this phenomenon based on statistically valid numbers of patients in various age groups are warranted. An exhaustive investigation at this time may be quite difficult, but with significant improvement in strain gage transducers, as recently advocated by Deck *et al.* (3), it may be possible in the near future.

In the case presented here, a significant increase of intracarotid pressure during angiography probably occurred but this was a special case with associated pathologic vascular physiology and cannot be compared to the studies of Greitz, and Bakay and Sweet. The peculiar circumstances here were an inadvertent selective internal carotid artery injection of 10 ml of contrast material into a markedly spastic internal carotid artery with associated spasm of the anterior and middle cerebral arteries. These conditions contributed to what we feel represented a wedge injection into a relatively inelastic, spastic arterial tree producing a significant increase in intra-arterial pressure with resultant aneurysmal rupture and spontaneous visualization of the opposite middle cerebral and supraclinoid internal carotid arteries. The magnitude of rupture into the right lateral ventricle was such that, within three seconds of injection, the entire ventricular system and a portion of the cervical subarachnoid space were opacified. The intraluminal pressure required to produce such a forceful rupture is unknown to us, but Turner (17) states that wedged forceful hand injections of 3–4 ml of contrast material into abnormal pulmonary arteries produces intraluminal pressures of greater than 800 mm Hg. It is, therefore, not inconceivable that the intracarotid pressure in our patient approached or even exceeded the 250–500 mm Hg range.

The aneurysmal rupture presented here was likely iatrogenic in nature, but its occurrence could have been prevented only by a preliminary low-volume, low-pressure test injection for needle placement and subsequent recognition of the

marked spasm of the internal carotid artery. It has not been our policy to routinely perform such test injections in approximately 1,500 carotid angiographies per year. Carotid angiography was performed in this patient to confirm or exclude the presence of an epidural or subdural hematoma and not for evaluation of subarachnoid hemorrhage. In view of subsequent findings, the initial presenting clinical picture can best be explained by subarachnoid hemorrhage and intracerebral hematoma formation secondary to spontaneous aneurysmal bleeding.

Radiographic demonstration of the rupture of congenital aneurysm implies a grave prognosis. All cases (5, 7, 8–10, 13) known to us, with a single exception (18), ended in death shortly thereafter, but in view of the rare occurrence of this phenomenon, the benefits of indicated cerebral angiography far outweigh this possible hazard.

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